

# The Basis of Predictive Modelling for Estimating Yield Loss and Planning Potato Cyst Nematode Management\*

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**Abstract:** The basis of modelling yield loss and population dynamics relations of potato cyst nematodes is that both are strongly density-dependent. Potato cyst nematodes (PCN; *Globodera pallida* and *G. rostochiensis*) are particularly suitable for analysing such relationships because they have only one generation per year, potato is their only field host, the juvenile nematodes within the egg are very durable (up to 20 years persistence), and they hatch mainly in response to specific chemicals exuded from host roots. Small populations increase the most, up to 50-fold, when a potato crop is grown. Multiplication rates decrease as the population density increases because damage decreases root system size and increases competition so that very large populations may actually be decreased when potatoes are grown. The newly formed eggs have a 'half-life' of c. two years when non-host, rotational crops are being grown.

Control is achieved largely by the use of rotation, the application of nematicidal chemicals, and growing resistant cultivars. As rotations are shortened, so PCN populations will be increased, and crop damage becomes more likely. Thresholds for damage vary with both soil type (greatest on sandy soils) and potato cultivar. Cultivars differ in their tolerance of PCN damage depending on how vigorously they grow, on their root sensitivity to damage from the PCN juveniles which invade close to root tips, on cultivar resistance which decreases the parasitic effect by reducing the numbers of developing PCN females, and on various environmental factors such as the amounts of fertiliser applied. The dependence of the yield-loss relationship on population density, soil type and cultivar effects has been described in a simple equation and assessed using field-trial data. Effects on yield are described in proportion to the PCN-free yield but the addition of information on expected yield (in tonnes ha<sup>-1</sup>) in the absence of PCN renders this equation predictive.

Nematicides are widely applied to infestations of potato cyst nematodes, both to prevent the crop from being damaged and to prevent population increase which could hazard the next potato crop in the rotation, but they are generally more effective at preventing the former than the latter.

A complex equation has also been developed to model the population dynamics of PCN. This equation incorporates a factor for host-crop growth and tolerance (from the yield-loss equation) and also the effects of host resistance. This latter is particularly relevant to *G. pallida*, where all the resistance currently available is determined by minor genes and hence is 'quantitative' or 'partial'. Effects of rotation and of nematicides can also be incorporated into this model. To provide a realistic prediction also requires accurate information on PCN population densities, species composition and distributions, and rates of PCN population decline between potato crops.

**Key words:** resistance, tolerance, integrated control, pesticides, rotation, prediction, damage, population dynamics.

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## 1 INTRODUCTION

Potato cyst nematodes (PCN; a complex consisting of *Globodera pallida* (Stone) and *G. rostochiensis* (Woll) are the most serious pests of potatoes in the UK; 22 000 ha of potatoes are treated with nematicides annually at a cost of c. £M7–8.<sup>1</sup> These carbamate and organophosphorus nematicides act on acetylcholine esterase, preventing the juvenile PCN from finding and invading host roots. They are water-soluble, non-phytotoxic and are applied at planting. Fumigant nematicides are also available but are little used in the UK. The decision to apply a nematicide is often based on a soil sample taken to provide an estimate of the pre-planting population density ( $P_i$ ) of PCN. A very general estimate that yield is decreased by two tonnes  $\text{ha}^{-1}$  for every 20 PCN eggs  $\text{g}^{-1}$  soil is used to decide whether treatment is likely to be cost-effective.<sup>2</sup> However, wider experience suggests a curvilinear relationship between PCN population density and yield (Fig. 1). Also, field trials have shown that the slope of the curve is influenced by environmental factors, including soil type, and by differences in cultivar tolerance of damage.<sup>1</sup> Consequently, the yield response to nematicides is much more variable than suggested above and a more realistic model, which allows for the interactions between PCN and other factors, is needed.

The pre-planting estimate of PCN population density also provides a basis for estimating population increase. As with damage, PCN multiplication rates are also density-dependent, like crop yield, decreasing with increasing density. This is because of increased juvenile invasion which slows the rate of root growth and hence increases competition between invading juveniles for feeding sites. A consequence of this is that a progressively decreasing proportion of juveniles becomes female. Hence, a second benefit from applying nematicides is to decrease the post-harvest population density of PCN but, because of the density-dependent effect on multiplication rate (Fig. 2), high levels of control are required to reduce large populations.

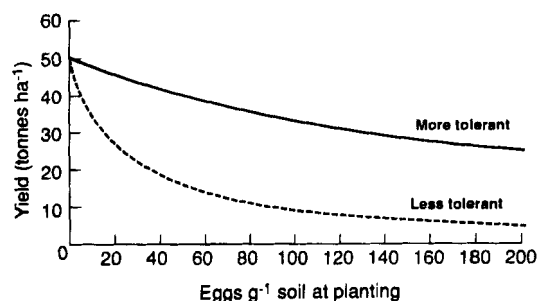


Fig. 1. The relationship between increasing population density of potato cyst nematodes at planting and yield of two potato cultivars with different degrees of tolerance.

Rotation is another means of controlling PCN; as they attack only potato (and tomato) the population declines when other crops are grown. However, PCN is very persistent, populations decreasing in the absence of a host by about 30% per annum, and viable infestations may persist for more than 20 years. Consequently, with short rotations (<4–6 years) many growers have suffered crop losses and have had to resort to additional control measures.

Resistant cultivars (which decrease or prevent PCN multiplication) are the third main means of controlling populations and have proved very effective against *G. rostochiensis*, all populations of which, in the UK, lack the virulence gene against the major gene for resistance (H1) derived from *Solanum tuberosum* L. ssp. *andigena* (CPC, 1673). This gene has been incorporated into a range of cultivars including Maris Piper which have been grown widely, often on short rotations. Unfortunately, *G. pallida* is completely virulent against the H1 gene and it has replaced *G. rostochiensis* as the dominant species in many potato fields. There is a very limited choice of cultivars with resistance to *G. pallida*. The few released so far have quantitative or polygenic resistance and do not provide the same high level of control given against *G. rostochiensis* by the H1 gene. So far, no major gene resistance has been found to *G. pallida*. The main source of quantitative resistance to *G. pallida* has been incorporated from the wild diploid species *Solanum vernei* Bitt. et Wittm. Because of the polygenic nature of this resistance, potato breeders have found it difficult to achieve >80% resistance whilst incorporating all the other commercial qualities needed. Another difference between *G. rostochiensis* and *G. pallida* is that the latter is much more variable and heterogeneous, which may be the result of there having been several different gene pools introduced into the UK.<sup>3</sup>

The widespread replacement of *G. rostochiensis* by *G. pallida* has created a much more serious problem, since *G. pallida* is more difficult to control because it appears to have a longer period of hatch and invasion than *G.*

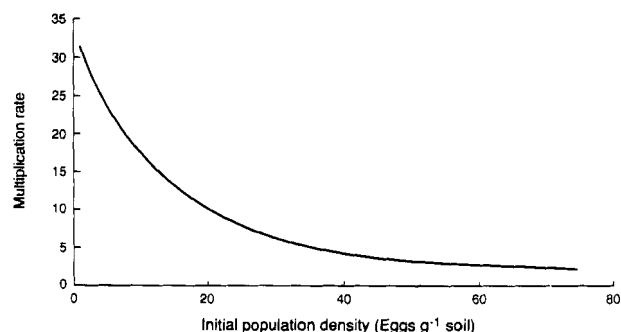


Fig. 2. Relationship between multiplication rates of potato cyst nematodes on a susceptible potato cultivar and population density at planting.

*rostochiensis* and hence is less well controlled by non-fumigant nematicides whose concentration in the soil diminishes rapidly. This problem is compounded by the lack of strong resistance to *G. pallida*.

The trend to shorter rotations, increased concentration of potato production on large units and on the more favourable land, and the increasing incidence of *G. pallida* all combine to produce a trend towards increasing problems. Coupled with this is the increasing awareness that chemical pesticides may have undesirable side-effects and that nematicides are amongst the most toxic chemicals used in agriculture. Hence, there is increasing pressure for them to be used as sparingly as possible and it is against this background that strategies for modelling the population dynamics of PCN, and effects on potato yields, have been developed.

## 2 BIOLOGY OF PCN

The two species of PCN do not interbreed, but they have similar biologies and life cycles. They co-evolved with *Solanum* spp. in South America and were accidentally introduced into Europe, probably in the nineteenth century. The original introductions have now been spread widely so that, in the UK, the majority of arable land which has grown potatoes frequently is now infested.

The cyst is formed from the dead body of the female which becomes tanned and contains up to 400 eggs. The dormant, second-stage juveniles within the eggs are resistant to drying and so the PCN is readily spread by wind and water and in soil adhering to machinery and seed tubers. A small proportion of eggs hatch spontaneously each spring but the majority require the stimulus of specific 'hatching factors' in exudate from host roots, when c.80% of the juveniles will hatch. A small proportion will still not hatch and are termed the 'carry-over'. Those juveniles that hatch do so over about a six- to eight-week period and use their hollow mouth stylet to bore into the potato root close to the tip. The mechanical damage they cause stunts root growth and affects nutrient uptake.<sup>4</sup> Within the root, the juveniles settle to feed close to the stele. The cell immediately adjacent to the head, and those adjoining it, become modified to form a 'syncytium' composed of a group of interconnected cells with enlarged, polyploid nuclei, dense cytoplasm and cell walls modified for increased solute transport.<sup>5</sup> The stimulus for these changes is secretions from nematode gland cells injected via the stylet. Those juveniles with large syncytia become female while those with smaller syncytia tend to become male.<sup>6</sup> The females swell and become balloon-shaped so that their bodies protrude on the outside of the root. After fertilisation in early July the females form eggs within their bodies and, when full, die and form cysts.

Damage is caused both by the invading juveniles, and by the parasitic feeding effect of the developing females. As resistant cultivars of potatoes produce hatching factor they hatch the juvenile PCN and suffer damage. However, because they produce small or degenerate syncytia, few juveniles are formed and the juveniles mainly become male or fail to develop. Consequently, some resistant cultivars are more tolerant of PCN damage than equivalent susceptible cultivars (because the parasitic females do not develop on them) but others suffer greater juvenile invasion damage and hence, are more intolerant.<sup>7</sup>

## 3 MODELLING PCN DAMAGE

The ability to predict yield losses from nematode damage is an essential requirement for making management decisions. This process is relatively simple for PCN because it has only one generation per year and yield losses can therefore be directly related to the pre-planting population density ( $P_i$ ). Most of the modelling has been based on single, simple equations describing the basic nature of the relationship.<sup>8</sup> Based on a competition equation, the relationship between proportional yield loss ( $y$ ) and  $P_i$  is described in terms of a tolerance population threshold ( $T$ ) below which no damage occurs, a minimum yield ( $m$ ) expressed as a proportion of the nematode-free yield obtained when all the available space in the roots is occupied by nematodes, and a constant slope factor ( $z$ ).<sup>9</sup>

$$y = m + (1 - m)z^{P_i - T} \text{ for } P_i > T, \text{ and } y = 1 \text{ for } P_i < T \quad (1)$$

When  $P_i$  is expressed on a logarithmic scale and the yield ( $y$ ) at a particular  $P_i$  is expressed as a proportion of the maximum yield in the absence of PCN, a sigmoidal curve is produced (Fig. 3). This relationship was validated by a series of glasshouse pot studies involving single plants and applied to total dry matter productivity. It does not, therefore, take account of the important interaction in the field when the tops of the

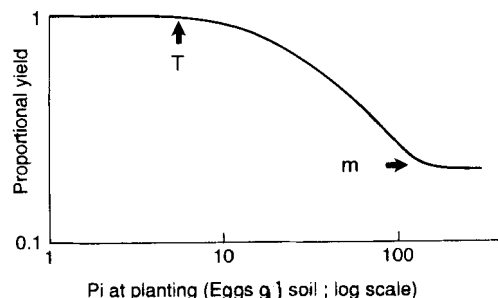


Fig. 3. Relationship between potato cyst nematode population density at planting (plotted on a logarithmic scale) and potato yields as a proportion of the nematode-free yield based on the Seinhorst equation.<sup>9</sup>

growing plants merge to form a continuous, light-intercepting canopy.<sup>1</sup> Equally, it does not allow for environmental factors to affect the value of  $z$ . Consequently, cultivar differences in tolerance of PCN damage have to be modelled through variations in  $m$ , which seems to be biologically inappropriate. Also  $T$  has proved to be too small (about two PCN eggs  $g^{-1}$  soil) to have practical value and it is difficult to devise accurate assessments of  $P_i$  at such low population densities.

Using data from a field trial where replicated plots were produced with a range of  $P_i$  values for *G. pallida*, an inverse linear model was proposed which was simpler than eqn. (1) but which described the data equally well.<sup>10</sup> If the value of  $m$ , the minimum yield, was fixed at zero (as experience indicates it can be for yields of potato tubers) only one parameter ( $c$ ) is needed rather than the three in eqn. (1), i.e.

$$y = 1/(1 + P_i/c) \quad (2)$$

However, since yield is still expressed as a proportion of the nematode-free yield, eqn. (2) cannot be used to predict actual yields. Also, effects of environmental factors and cultivar tolerance differences on the slope parameter ( $c$ ) can be determined only by experimentation.

A further series of replicated field trials was conducted involving cultivars with different tolerances to PCN damage. Because these trials were on sites with different soil types, it proved possible to partition the slope parameter  $c$  into a site (soil type) and a cultivar (tolerance) component ( $g$  and  $s$  respectively) (Table 1) (Phillips, M. S. & Hackett, C. A., unpublished results). Hence, if the maximum, nematode-free yield can be predicted, and light-interception/water-use models exist to do this, then a predictive model is possible.

#### 4 MODELLING PCN POPULATION DYNAMICS

The population dynamics of PCN are strongly density-dependent and an equation was developed based on a

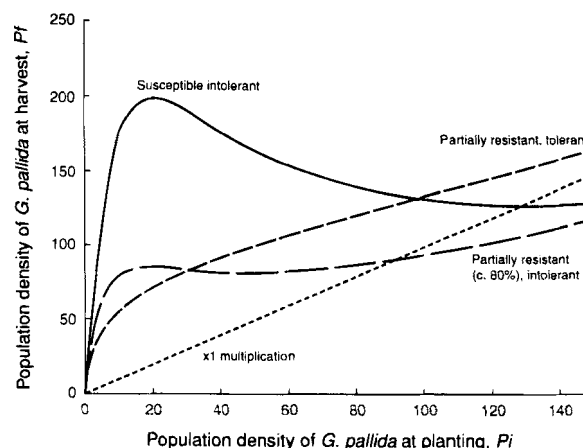


Fig. 4. Effect of pre-planting *G. pallida* populations ( $P_i$ ) on post-harvest populations densities ( $P_f$ ) on a fully susceptible, and on a partially resistant cultivar of potato which is either tolerant or intolerant of damage.

logistic model.<sup>11</sup> This equation includes the carry-over proportion ( $cp$ ) of the PCN population which does not hatch and the damage caused to the potato plant, but assumes this to be constant for all cultivars. Essentially, rates of population increase are greatest at low values of  $P_i$  and decrease as the  $P_i$  increases until, at the equilibrium density, the pre-planting and post-harvest populations are the same. Above the equilibrium density the population decreases (Fig. 4). As the rate of PCN multiplication is influenced by how well the host grows and by its degree of resistance, a modification was made to allow for cultivar tolerance differences and for various degrees of resistance.<sup>11</sup> This equation (not described here but described in Ref. 8) has been validated by field trial data (Phillips, M. S. & Trudgill, D. L., unpublished).

Multiplication rates of *G. pallida* on partially resistant cultivars mirror those on susceptible cultivars, but maximum rates of multiplication and equilibrium densities are proportionally decreased. Hence, on a cultivar with 80% resistance the maximum rate of multiplication (achieved only at a very low  $P_i$ ) will be decreased from  $c.50$ -fold to  $c.10$ -fold. Considerable differences in the virulences of different populations of *G. pallida* on the

TABLE 1  
Estimates of Tolerance Parameters<sup>a</sup>

Parameter <sup>b</sup>	Maris Piper/12380	Cultivar/clone Morag/Santé		Glenna
$g$	47.9 ( $\pm 8.53$ ) Intolerant	81.1 ( $\pm 13.1$ )		156.6 ( $\pm 34.8$ ) Tolerant
		Site		
$s$	Luffness 0.35 (0.11) Most damage	Terrington 1	Nocton 1.49 (0.27)	Latch 2.47 (0.61) Least damage

<sup>a</sup> Standard errors in parenthesis.

<sup>b</sup>  $g$ -cultivar parameter;  $s$ -site parameter.

same partially resistant cultivar, and interactions which affect rates of multiplication between some populations of *G. pallida* and some partially resistant cultivars (Phillips, M. S., unpublished) further complicate the modelling of *G. pallida* population dynamics.

The major conclusions relevant to the long term control of *G. pallida* are that partially resistant cultivars will still allow some population increase at low population densities, and that, at higher  $P_i$  values, tolerant cultivars (which still grow well) will increase *G. pallida* populations more than intolerant cultivars (Fig. 4). From this analysis it is also evident that nematicides will have their greatest effect in decreasing the rate of PCN multiplication when applied at low  $P_i$  (when there is little damage and loss of yield, and hence no economic return but when potential multiplication rates are at their greatest) rather than at high  $P_i$  values. However, a more environmentally acceptable strategy would be to combine reduced rates of nematicides with one of the available partially resistant cultivars since the effects of the two are additive.

## 5 INTEGRATED CONTROL OF PCN

For practical purposes, control of PCN multiplication depends upon rotation, use of nematicides and cultivar resistance. Crop damage is a consequence of previous management and hence is also determined by the same factors but is additionally affected by cultivar tolerance of damage and by various aspects of management (e.g. rates of fertiliser). These will also feed back into the population dynamics model, as already indicated, because the better the crop grows the more the PCN is likely to multiply.

Management of *G. rostochiensis* in the UK is relatively simple, as there is only one pathotype (Ro1) which is completely avirulent against the H1 resistance gene. Repeated growing of H1 resistant cultivars will progressively decrease the  $P_i$  (by c. 80% for each crop) and in longer rotations (>5 years) it will probably be possible to alternate resistant and susceptible cultivars without damage (Fig. 5). Where a small proportion of *G. pallida* is mixed with the *G. rostochiensis* the former will become the dominant species after three to four crops of a resistant cultivar. Alternating resistant and susceptible cultivars will slow this process.<sup>12</sup>

In the Netherlands attempts to grow starch potatoes on two-year rotations by integrating nematicides and resistant cultivars have encountered a range of difficulties. Pathotypes of *G. rostochiensis* virulent against the H1 gene are present, but selection of *G. pallida* has still been the major problem (Bakker, J., pers. comm.). Also, nematicide use (mainly fumigants which have to be applied at high rates) has reached unacceptable levels.

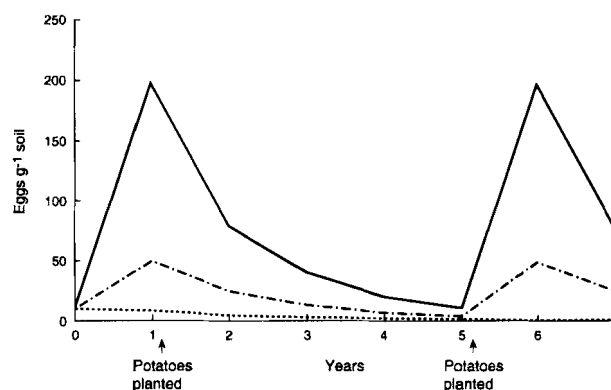


Fig. 5. Effects on initial soil population density of 10 eggs  $g^{-1}$  of potato cyst nematodes of growing (—) a susceptible, (---) a fully resistant and (-·-) a partially resistant cultivar (c. 80%) on a four-year rotation.

Once *G. pallida* becomes the major problem, control of PCN becomes more difficult. Experience suggests that *G. pallida* is less well controlled by non-fumigant nematicides than *G. rostochiensis*, and that non-fumigant nematicides are more effective on some soils than on others, partly due to enhanced rates of breakdown.<sup>13</sup> Consequently, at some sites where potatoes have been grown on short rotations, nematicides have failed to prevent significant crop loss. A few cultivars partially resistant to *G. pallida*, such as Santé, are available and, in conjunction with a nematicide, can provide adequate control in short rotations. However, Santé is the reluctant choice of many growers who wish to alternate it with more commercially acceptable, susceptible cultivars. Experience in the Netherlands has shown that in short rotations, nematicides will probably have to be applied to all potato crops, even those with partial resistance, and that such a strategy is not sustainable in the longer term.

## 6 THE FUTURE

An interactive, predictive model for PCN management which can be used on a personal computer, and which has an economic component, would enable growers and their advisors to anticipate the longer-term consequences on PCN population densities of their potato production practices. To be predictive, such a model would require an estimate of the PCN-free yield and more precise information on PCN population densities, distributions, species composition and virulence characteristics than most farmers currently possess. With the advent of satellite-guided positional information there may be opportunities for targeting control measures more precisely. In the short term, however, it is difficult to envisage intensive potato production continuing on land infested with *G. pallida* without considerable reliance on nematicides. Engineered resistance, particularly using novel genes, is a prospect in the medium term, but

the tolerance, uniformity of effect and consumer acceptability of such transgenic cultivars remain to be determined. Alternatives such as trap cropping may have a place in decreasing large populations of PCN, but control of PCN, and especially of *G. pallida*, depends on effective management designed to prevent large populations developing. To this end there is a shortage of commercially acceptable cultivars with good resistance to *G. pallida*.

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